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14. ABSTRACT

This paper reviews the roles of hot skin (>35°C) and body water deficits (>2% body mass; hypohydration) in impairing submaximal aerobic performance. Hot skin is associated with high skin blood flow requirements and hypohydration is associated with reduced cardiac filling, both of which act to reduce aerobic reserve. In euhydrated subjects, hot skin alone (with a modest core temperature elevation) impairs submaximal aerobic performance. Conversely, aerobic performance is sustained with core temperatures >40°C if skin temperatures are coolwarm when euhydrated. No study has demonstrated that high core temperature (-40°C) alone, without coexisting hot skin, will impair aerobic performance. In hypohydrated subjects, aerobic performance begins to be impaired when skin temperatures exceed 27°C, and even warmer skin exacerbates the aerobic performance impairment (-1.5% for each l°C skin temperature). We conclude that hot skin (high skin blood flow requirements from narrow skin temperature to core temperature gradients), not high core temperature, is the 'primary' factor impairing aerobic exercise performance when euhydrated and that hypohydration exacerbates this effect.

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Symposium Report

High skin temperature and hypohydration impair aerobic performance

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This paper reviews the roles of hot skin (>35°C) and body water deficits (>2% body mass; hypohydration) in impairing submaximal aerobic performance. Hot skin is associated with high skin blood flow requirements and hypohydration is associated with reduced cardiac filling, both of which act to reduce aerobic reserve. In euhydrated subjects, hot skin alone (with a modest core temperature elevation) impairs submaximal aerobic performance. Conversely, aerobic performance is sustained with core temperatures >40°C if skin temperatures are cool-warm when euhydrated. No study has demonstrated that high core temperature (~40°C) alone, without coexisting hot skin, will impair aerobic performance. In hypohydrated subjects, aerobic performance begins to be impaired when skin temperatures exceed 27°C, and even warmer skin exacerbates the aerobic performance impairment (–1.5% for each 1°C skin temperature). We conclude that hot skin (high skin blood flow requirements from narrow skin temperature to core temperature gradients), not high core temperature, is the 'primary' factor impairing aerobic exercise performance when euhydrated and that hypohydration exacerbates this effect.

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Heat stress impairs submaximal and maximal aerobic exercise performance (Sawka *et al.* 2011). For maximal intensity exercise, cardiovascular mechanisms related to oxygen delivery are likely to limit performance in the heat (Rowell *et al.* 1966; González-Alonso & Calbet, 2003). The mechanisms limiting sustained, submaximal intensity exercise in the heat include cardiovascular, CNS and metabolic (glycogen depletion) changes (Cheung & Sleivert, 2004; Sawka *et al.* 2011). Metabolic limitations are minor and specific to particular exercise tasks in the heat (Cheung & Sleivert, 2004). Cardiovascular mechanisms were historically assumed to be the primary factor impairing submaximal performance in the heat (Rowell, 1986), but the sustainment of skeletal muscle blood flow at exhaustion shifted the emphasis towards CNS limitations and the role of high core temperatures (Nielsen *et al.* 1990, 1993).

Bodil Nielsen and colleagues (Nielsen *et al.* 1990) proposed that a high core temperature (~40°C) 'having an effect on the CNS in reducing the motor drive for performance' is the critical factor impairing submaximal intensity aerobic performance in the heat. During the past decade, the 'critical' core temperature hypothesis has been widely attributed as the primary mechanism impairing submaximal aerobic performance in the heat. Deteriorated CNS function may contribute to impaired aerobic performance in the heat (Nybo & Nielsen, 2001*a*), but the importance of a high core temperature has rarely been questioned. Studies supporting the 'critical' core temperature hypothesis have simultaneously induced high core temperatures with hot skin (Nielsen *et al.* 1990, 1993; González-Alonso *et al.* 1999). To our knowledge, no study has demonstrated that high core temperature alone will impair aerobic performance.

This paper reviews recent evidence that hot skin (>35°C) alone can impair submaximal aerobic performance. Additional evidence will be provided

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Table 1. Estimated whole-body skin blood flow (SkBF) requirements* during prolonged, severe running exercise† at different body core (T_c) and skin temperatures (T_{sk})

T_c (°C)	T_{sk} (°C)	Gradient (°C)	SkBF (l min^{-1})
38	30	8	1.1
38	32	6	1.5
38	34	4	2.2
38	36	2	4.4
39	30	9	1.0
39	32	7	1.3
39	34	5	1.8
39	36	3	2.9

*Equation for skin blood flow: $Q_s = 1/C \times h/(T_c - T_{sk})$, where C is the specific heat of blood (~0.87 kcal °C^{-1} l^{-1}), h the heat production (in kcal min^{-1}) and Q_s the skin blood flow (Rowell, 1986). † Net heat production (7.7 kcal min^{-1}) estimated using 60 kg body mass and 325 m min^{-1} running velocity (approximate pace for men's world class 42 km footrace) after subtracting for work (20% efficiency) and 50% dry and evaporative heat losses.

that if skin temperatures (T_{sk}) are cool-warm, aerobic performance can be sustained despite high core temperatures (T_c). Hot skin narrows the T_{sk} to T_c gradient, which increases skin blood flow requirements (Rowell, 1986) and may be the 'primary' factor impairing submaximal aerobic exercise performance in the heat. Body water deficits (>2% body mass; hypohydration) will exacerbate the effect by reducing central blood volume. We therefore postulate that during exercise heat stress, hot skin and hypohydration act in concert to reduce aerobic reserves, which increases the relative exercise intensity and perception of effort.

Physiology of skin temperatures and hypohydration

During exercise in the heat, the most significant physiological burden is to support high skin blood flow for heat dissipation (Sawka *et al.* 2011). Skin temperature is elevated in proportion to ambient temperature and humidity (Gagge & Gonzalez, 1996), while T_c is elevated in proportion to exercise intensity and is largely independent of the environment during compensable heat stress (Sawka *et al.* 2011). Warm-hot skin is associated with a greater skin blood flow and cutaneous venous compliance, which augments cardiovascular strain (Sawka *et al.* 2011). For this review, we define hot skin as 35°C and above, warm skin as 30–34.9°C and cool/cold skin as <30°C. We recognize that skin temperature effects are a continuum and that the T_{sk} to T_c gradient alters these relationships.

Table 1 illustrates the effects of different T_{sk} and T_c combinations on estimated (Rowell, 1986) whole-body skin blood flow requirements during combined exercise and heat stress. An elevated T_{sk} increases skin blood flow at any given T_c, while an elevated T_c reduces skin blood flow requirements at any given T_{sk}. The rows beginning with T_c 38 and 39°C highlight an often unappreciated point; at any given skin temperature, an elevation in core temperature reduces whole-body skin blood flow and can be viewed as a positive response for sustaining aerobic performance in the heat. For example when comparing T_c of 39°C to a T_c of 38°C at equivalent T_{sk} of 36°C, SkBF is reduced from 4.4 to 2.9 l min^{-1}. Figure 1 demonstrates the impact of warm-hot T_{sk}, at constant T_c (~37.5°C), on cardiovascular strain during light-intensity (metabolic rate ~450 W) exercise (Cheuvront *et al.* 2003). The heart rate (HR) elevation during exercise was an exponential function of skin warming beyond T_{sk} ~35°C. The high skin blood flow requirements act to reduce cardiac filling and elevate HR for a given cardiac output (Trinity *et al.* 2010; Stohr *et al.* 2011*b*). Conversely, rapidly cooling T_{sk} has a profound effect on reducing HR and sustaining mean arterial pressure during exercise in the heat (Shaffrath & Adams, 1984). In addition, hot skin can be associated with reduced cerebral blood flow and cerebral oxygen delivery during moderate-intensity exercise (Nybo & Nielsen, 2001*b*; Nybo *et al.* 2002; Rasmussen *et al.* 2010).

During combined exercise and heat stress, hypohydration augments hyperthermia and cardiovascular strain in proportion to the magnitude of body water deficit (Sawka *et al.* 1985). Hypohydration reduces cardiac filling (Stohr *et al.* 2011*b*) and stroke volume during combined exercise and heat stress, making it difficult to maintain cardiac output (Montain & Coyle, 1992) and sustain muscle blood flow when heat stress is severe (González-Alonso *et al.* 1998).

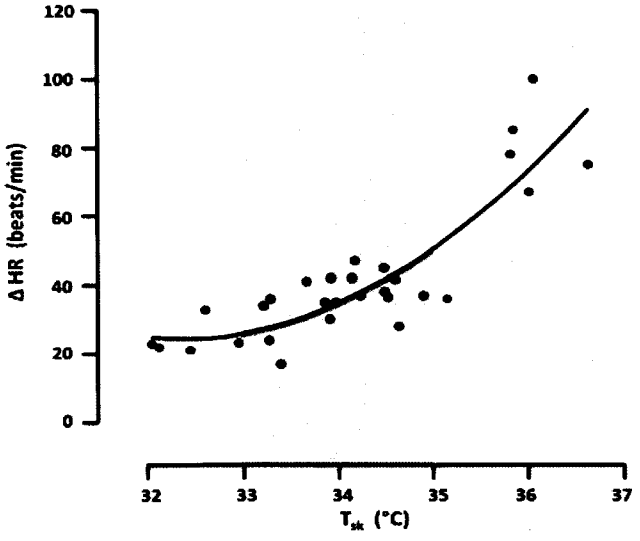

Figure 1. Impact of high skin temperature, with a constant core temperature, on elevating heart rate during light-intensity exercise (metabolic rate ~450 W)
From Cheuvront *et al.* (2003).

Aerobic performance

Heat stress. Warm-hot T_{sk} degrades maximal aerobic power ($\dot{V}_{O_2max}$) in proportion to the T_{sk} elevation (Arngrimsson *et al.* 2003). Thus, when performing exercise at a given metabolic rate, a person with warm-hot skin will work at a greater percentage $\dot{V}_{O_2max}$ compared with temperate conditions. Figure 2 demonstrates that marathon race performance is progressively slower with increased environmental (wet bulb globe temperature; WBGT) heat stress (Ely *et al.* 2007). Skin temperature is elevated with WBGT (Gagge & Gonzalez, 1996), but T_c may or may not be elevated, as it depends upon the sustainment of exercise intensity and heat exchange biophysics (Sawka *et al.* 2011). Therefore, marathon race performance might slow as a function of elevated T_{sk}.

Laboratory studies consistently demonstrate that T_{sk} elevations impair submaximal intensity aerobic performance. González-Alonso *et al.* (1999) employed a time-to-exhaustion (TTE; 60% $\dot{V}_{O_2max}$) test, during which subjects wore a water-perfused suit. When T_{sk} was elevated from ~36 to 38°C, TTE was shortened from 56 to 31 min; however, between the two trials, T_c (~40°C) and HR (~188 beats min^{-1}) were similar at exhaustion. MacDougall *et al.* (1974) used a similar combination of TTE test (70% $\dot{V}_{O_2max}$) with water-perfused suit to show that when T_{sk} was raised incrementally from ~29 to ~32 and then ~35°C, TTE was shortened from 90 to 75 and then 48 min, respectively, despite similar core temperatures at exhaustion (~39.5°C). Tatterson *et al.* (2000) used a time-trial (TT) test and reported that performance was impaired by ~6% in a warm environment when T_{sk} was ~33°C, *versus* 27°C in a temperate environment. Core temperature and HR levels were again similar at exhaustion (~39.3°C and ~195 beats min^{-1}), as in other studies. Most recently, Periard *et al.* (2011) reported a ~13% decrement in mean power output during a 40 km TT in hot *versus* temperate environmental conditions that produced T_{sk} of 36 and 28°C, respectively. Although T_c was higher at exhaustion in the heat (39.8 *versus* 38.9°C), pacing strategy fell off significantly after 20 min of cycling when T_c was similar in both trials (~38°C), while T_{sk} was already >5°C higher in the heat.

Performance studies cited as directly supporting the 'critical' core temperature hypothesis have simultaneously elicited high core temperatures with hot skin. In the original study of Nielsen *et al.* (1993), subjects completed a TTE test (60% $\dot{V}_{O_2max}$) for 9–12 consecutive days in a hot environment as part of a heat acclimation experiment. Heat acclimation increased TTE from 48 to 80 min over the test days, with exhaustion consistently coinciding with T_c ~40°C and T_{sk} ~37°C. González-Alonso *et al.* (1999) manipulated initial body temperatures prior to a TTE test (60% $\dot{V}_{O_2max}$) in a hot environment by applying precooling, no precooling and preheating to subjects. The critical core temperature explanation for fatigue resulted from exhaustion coinciding with a consistently high T_c (~40°C), but T_{sk} (~37°C) and HR (~196 beats min^{-1}) were equally consistent, with the HR near maximal levels based on age.

There is evidence that hot T_{sk} (>35°C) alone can degrade aerobic performance. Ely *et al.* (2010) measured the impact of two environmental conditions (40 and 20°C) on a 15 min TT performance test where T_c elevation was modest and similar in both trials (~38.2°C), but the compensable environments produced cool-warm (30°C) or hot skin (36°C). Time trial performance was impaired by 17% with hot T_{sk}, although a similar HR (~180 beats min^{-1}) was achieved. These findings are consistent with studies employing uncompensable heat stress to produce hot T_{sk} (>35°C) while performing a walking TTE test (Sawka *et al.* 1992; Montain *et al.* 1994; Latzka *et al.* 1998). During those studies, physical exhaustion routinely occurred (~50% of cases) at relatively low T_c (<38.5°C), but with high HR relative to the exercise intensity. Therefore, hot skin will impair performance and induce exhaustion well below levels associated with the 'critical' T_c hypothesis.

There is evidence that competitive running performance (velocity) can be preserved despite high T_c ≥40°C, if T_{sk} is cool-warm (Ely *et al.* 2009; Lee *et al.* 2010). Ely *et al.* (2009) had highly trained runners perform an 8 km running TT on a 400 m track in compensable environmental conditions eliciting cool-warm T_{sk} (32–34°C). They measured running

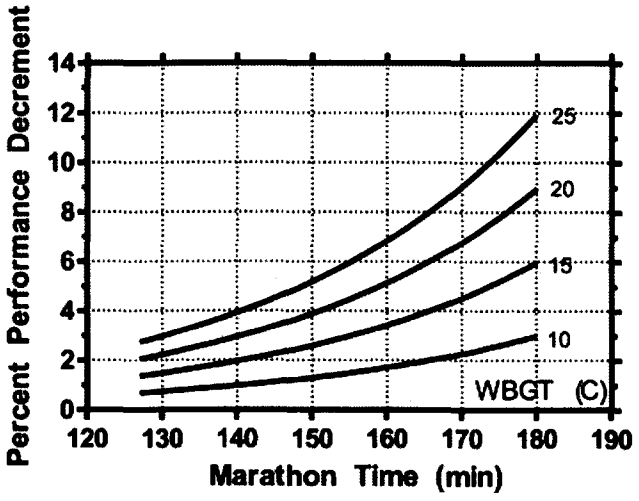

Figure 2. Performance decrement (*y*-axis) based on marathon finishing time (*x*-axis) with increasing wet bulb globe temperature (WBGT)
This nomogram was developed by analysing weather data and finishers from seven marathons over many years (Ely *et al.* 2007).

velocities over 200 m segments and found no difference when T_c was below (first 6.5 km) or above 40°C (final 1.5 km). Figure 3 presents the individual data for average running velocities when T_c was below and above 40°C. Lee *et al.* (2010) also examined running performance in a warm environment during a longer, 21 km race, in which velocitiy was determined for 3 km intervals. They too found that high core temperature (≥39.5°C) was common and not associated with reduced performance. Although T_{sk} was not measured, the T_{sk} prediction equation of Adams (1977) for outdoor running in the sun would suggest a value near 32°C.

Hypohydration. Hypohydration impairs maximal aerobic power in hot environments (Craig & Cummings, 1966) and submaximal aerobic performance in temperate and warm-hot environments (Cheuvront *et al.* 2005; Castellani *et al.* 2010; Kenefick *et al.* 2010). The following studies demonstrated that hypohydration impairs submaximal aerobic performance and that the impairment is augmented by high T_{sk}. In those studies, T_c was <39°C and therefore well below the 'critical' T_c.

Cheuvront *et al.* (2005) tested the effect of hypohydration on aerobic performance using a 30 min exercise preload at ~50% $\dot{V}_{O_2max}$, followed by a 30 min TT in temperate and cold environments. Hypohydration by 3% body mass impaired performance by 8% in the temperate (T_{sk} ~29°C) but not in the cold environment (T_{sk} ~20°C). Castellani *et al.* (2010) used a nearly identical test whereby T_{sk} was ~32°C in both hypohydration and euhydration trials. Hypohydration by 4% body mass impaired performance by 18%. Kenefick *et al.* (2010) further characterized the interaction between environmental conditions and hypohydration by having subjects exercise for 30 min (50% $\dot{V}_{O_2max}$) followed by a 15 min TT in 10, 20, 30 and 40°C environments (inducing stepwise increases in T_{sk} from 26 to 36°C) when euhydrated and when hypohydrated by 4% body mass. Hypohydration impaired aerobic performance by 12 and 23% when T_{sk} was 33 and 36°C, respectively.

Figure 4 plots the impact of hypohydration on aerobic performance from the preceding three studies (Castellani *et al.* 2010; Cheuvront *et al.* 2005; Kenefick *et al.* 2010). These studies employed similar procedures over a broad range of T_{sk} from 20 to 36°C. Segmented regression (Vieth, 1989) was used to approximate the statistical T_{sk} threshold for performance impairment using individual study data points ($n = 53$ paired observations). The threshold which best minimized the residual sums of squares was shown to be 27.3°C, and warmer skin accentuated the performance impairment by ~1.3% for each additional 1°C rise in T_{sk} similar to that reported by Kenefick *et al.* (2010).

High skin temperature/relative intensity hypothesis

Cheuvront *et al.* (2010) proposed that impaired submaximal aerobic performance in the heat might be explained by warm-hot T_{sk} reducing $\dot{V}_{O_2max}$. A large $\dot{V}_{O_2max}$ is a prerequisite for success in sports where

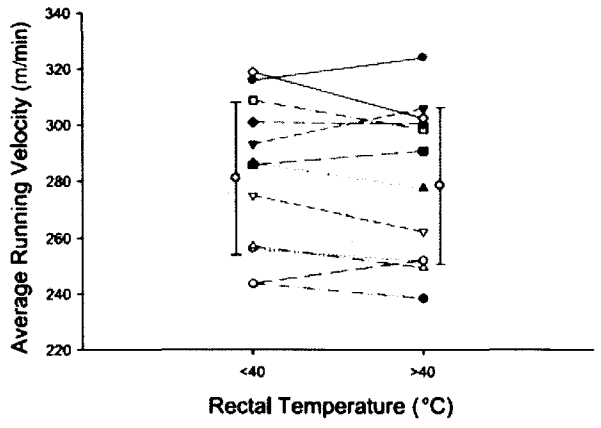

Figure 3. Time trial running velocities of 12 highly trained runners in compensable environmental conditions (cool-warm skin temperatures) when their core temperatures were below (mean of first ~32 200 m segments) or exceeded a core temperature of 40°C (mean of last approximately eight 200 m segments)
From Ely *et al.* (2009).

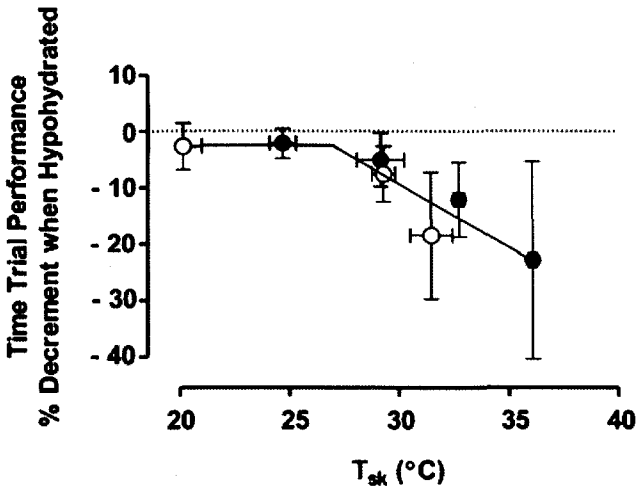

Figure 4. Percentage decrement in submaximal aerobic performance from euhydration as a function of skin temperature (T_{sk}) when hypohydrated by 3–4% of body mass
Data are means (error bars are 95% confidence intervals) compiled from three studies (Cheuvront *et al.* 2005; Castellani *et al.* 2010; Kenefick *et al.* 2010) employing similar experimental procedures and time trial (TT) performance tests. Filled circles represent 15 min TT tests; open circles represent 30 min TT tests. At a T_{sk} intercept of ~27°C, the percentage decrement in aerobic exercise performance declines linearly by ~1.3% for each 1°C rise in T_{sk}, similar to the single study of Kenefick *et al.* (2010) The best-fit equation for the second linear line segment is $y = -1.26x + 26.37$.

aerobic metabolism predominates (Bassett, & Howley, 2000), and $\dot{V}_{O_{2max}}$ is reduced incrementally with warm-hot T_{sk} (Arngrimsson *et al.* 2003). When $\dot{V}_{O_{2max}}$ is reduced, the resultant increased percentage $\dot{V}_{O_{2max}}$ results in impaired submaximal exercise capacity (Gleser & Vogel, 1973*a*,*b*). If relative exercise intensity is increased, constant-rate exercise (TTE) will be more difficult to sustain (earlier fatigue) or require a slowing of self-paced exercise (TT) to achieve a similar sensation of effort. An increased percentage $\dot{V}_{O_{2max}}$ is associated with greater cardiopulmonary stress (HR and respiration) and elevated perceived exertion, while warm-hot skin is associated with elevated thermal discomfort (Gagge *et al.* 1969; Gonzalez & Gagge, 1973). Other physiological cues being sensed might include cardiopulmonary baroreceptor unloading (Stohr *et al.* 2011*a*,*b*), reduced cerebral perfusion or cerebral oxygenation (Rasmussen *et al.* 2010) and arterial hypocapnia (Sawka *et al.* 1980). Likewise, if CNS function is deteriorated by heat stress and contributes to impaired performance, the afferent input from skin (Kunsch *et al.* 1995), muscle (Todd *et al.* 2005) and perhaps osmoreceptors/baroceceptors when hypohydrated (Montain & Tharion, 2010) might all contribute to altering the signal processing.

We conclude that: (1) hot skin (high skin blood flow requirements from narrow T_{sk} to T_c gradients), not high core temperature, is the 'primary' factor impairing submaximal aerobic performance when euhydrated; (2) hypohydration impairs submaximal aerobic performance when skin temperature is ~27°C, and even warmer skin exacerbates (–1.5% for each 1°C T_{sk}) these decrements; and (3) high core temperature (~40°C) alone does not impair aerobic performance.

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